
Appendix G

TRE Case Study: Linden Roselle Sewerage Authority, New Jersey

Abstract

TRE Goal:	96-hour LC50 \geq 50% Interim goal of LC50 \geq 30%
Test Organism:	<i>M. bahia</i>
TRE Elements:	Facility performance evaluation, TIE, toxicity source evaluation
Toxicants Identified:	Ammonia, non-polar organic compounds, surfactants
Toxicity Controls:	Pretreatment limits

Summary

Ammonia was confirmed as the primary cause of toxicity, and pretreatment limits were developed to reduce effluent ammonia concentrations. Secondary causes of toxicity were complex and highly variable. Toxicity-based procedures were used to identify industrial sources of toxicity and develop pretreatment limits to control secondary causes of toxicity.

In 1997, a major source of ammonia was eliminated. An acute toxicity test performed since then showed a reduction in effluent toxicity (LC50 = 72%) to compliance levels (i.e., LC50 $>$ 50%). Additional tests are planned to confirm this initial result.

Key Elements

1. TIE procedures may need to be modified to evaluate multiple causes of effluent toxicity. In this study, it was necessary to remove toxic effluent concentrations of ammonia in the TIE before other causes of toxicity could be identified and confirmed.
2. If TIE analyses are successful in confirming causes of effluent toxicity (e.g., ammonia), chemical-specific analyses can be used to identify

sources and pretreatment limits can be developed for controllable toxicants.

3. If the TIE is inconclusive or the causes of toxicity are variable and complex, the RTA approach can be used to track the industrial sources of toxicity in the collection system. Once identified, the toxic dischargers can be required to meet pretreatment limits for toxicity.
4. If effluent toxicity is contributed by controllable industrial sources, pretreatment controls are more practical than in-plant controls.

Introduction

Permit Requirements

The LRSA New Jersey Pollutant Discharge Elimination System (NJPDES) permit contains an acute whole effluent toxicity limit of LC50 $>$ 50% effluent. A 96-hour static renewal *M. bahia* (mysid) test is used to monitor compliance with the limit. Based on observed toxicity to mysids, the NJPDES permit was amended to include a requirement to perform a TRE. In July 1992, the LRSA entered into an administrative consent order (ACO) with the New Jersey Department of Environmental Protection (NJDEP) to establish a compliance schedule for reducing acute effluent toxicity. The ACO established a compliance date of October 31, 1996, if pretreatment controls are implemented and a compliance date of December 31, 1997, if in-plant controls are implemented. The ACO also includes TRE milestones and an interim whole effluent toxicity limitation of an LC50 of 30%. The acute effluent toxicity limit of an LC50 of 50% becomes effective on May 1, 2000.

Description of the Treatment Plant

The LRSA POTW serves a 13-square-mile area in northeastern New Jersey. The POTW has a design

flow of 17 mgd and is presently treating a wastewater flow of about 13 mgd. Approximately 20% of the influent flow is contributed by 40 industrial users. Primary treatment consists of screening and degritting followed by primary sedimentation. The primary effluent is then treated by roughing (trickling) filters and conventional activated sludge treatment. Following secondary clarification, the effluent is disinfected with chlorine and then discharged to the Arthur Kill estuary. The NJPDES permit specifies that samples for toxicity testing be collected prior to chlorination.

Plant Performance Evaluation

A limited POTW performance evaluation was conducted during a USEPA TRE research study to determine if POTW operations or performance was contributing to the observed acute toxicity. The POTW performance evaluation findings showed that industrial wastewater contributions have a significant effect on the variability and concentration of influent constituents. For example, in 1987, influent BOD₅ varied from 292 to 636 mg/L, oil and grease ranged from 11 to 132 mg/L, and ammonia-nitrogen varied from 17 to 119 mg/L (Morris et al., 1990). The influent variability requires the LRSA to make significant modifications to plant operations, such as operating one or two aeration basins, to maintain optimum treatment. Despite this variability, the LRSA has consistently met NJPDES permit effluent limits for conventional pollutants.

Overall, the POTW performance evaluation indicated that the operation and performance of the LRSA POTW was satisfactory and the treatment processes did not appear to be contributing to effluent toxicity (Morris et al., 1990). The POTW performance evaluation also indicated that the ammonia concentrations observed in the effluent warranted further evaluation as a cause of effluent toxicity.

Pretreatment Program Review

Monthly average influent ammonia concentrations at the LRSA have been as high as 150 mg/L. A review of the influent ammonia data indicated consistently lower ammonia levels in July of each year (LRSA, 1990a). The decreased ammonia concentrations were related to the temporary shutdown of a manufacturing process at a major industrial contributor.

Toxicity Identification Evaluation

An objective of the LRSA TRE was to identify the causes of effluent toxicity in order to select controls for

reducing toxicity. Initial TIE Phase I and Phase II testing was performed in 1989 using *C. dubia* as a surrogate test species. *C. dubia* were used because little information was available at the time for using mysids as a TIE test organism. Subsequent TIE testing in 1991 was performed using mysids to confirm that the causes of toxicity identified using *C. dubia* were also causes of toxicity to mysids.

TIE Phase I

During the USEPA study, three effluent samples were tested using the TIE Phase I procedures (USEPA, 1988). The Phase I results and ammonia data indicated that ammonia was a primary cause of effluent toxicity. Toxicity reduction by C18 SPE suggested that non-polar organic compounds were also contributing to effluent toxicity (Morris et al., 1990).

TIE Phases II and III

TIE Phase II (USEPA, 1989b) and Phase III (USEPA, 1989c) analyses were performed using *C. dubia* and mysids to identify and confirm ammonia and non-polar organic toxicants as causes of effluent toxicity (LRSA 1990b, 1991; Morris et al., 1992). It was necessary to remove ammonia toxicity in the TIE before other toxicants could be evaluated. A serial treatment approach was used to evaluate the contribution of non-polar organic toxicants to acute effluent toxicity. Effluent samples were first treated with zeolite to remove ammonia and then non-polar organic toxicity was evaluated using C18 SPE column treatment and GC/MS analyses. A separate C18 SPE column test was performed using whole effluent to determine if zeolite treatment had removed non-polar organic toxicity.

Results of the non-polar organic toxicant confirmation tests, presented in Table G-1, show that filtration, C18 SPE column treatment, and zeolite treatment reduced toxicity to both mysids and *C. dubia*. The combined treatment steps removed all of the acute toxicity to both species. Following filtration, zeolite treatment removed 1.3 to 2.0 TUa, while the C18 SPE column removed 1.5 to 4.3 TUa. Acute toxicity to both species was recovered in the 80 to 100% methanol/water fractions from the C18 SPE column. Although only 0.3 TUa were recovered from the column, previous tests had shown greater recovery (>2 TUa). The lower recovery of non-polar organic toxicity in this sample may be due to the presence of toxicants that are difficult to elute from the C18 SPE column (e.g., surfactants were indicated as a possible toxicant based

Table G-1. TIE Phase III Results: Non-Polar Organic Compound Confirmation (LRSA POTW)

Sample Description*	TUa (100/LC50)	
	<i>C. dubia</i> †	<i>M. bahia</i> ‡
Baseline toxic units	4.3	8.5
Post-filtration treatment	2.8	6.3
Aliquot No. 1		
Post-filtration and C18 SPE column treatment (original pH)	100 §	100 §
Combined toxic methanol/water C18 SPE column fractions#	0.3	0.3
Aliquot No. 2		
Post-zeolite treatment	1.5	4.3
Post-zeolite and C18 SPE column treatment	<1.0	<1.0
Combined toxic methanol/water fractions from zeolite/ C18 SPE column treatment#	0.3	0.3

* Effluents of serial treatment steps.

† 48-hour *C. dubia* acute toxicity test.

‡ 96-hour *M. bahia* acute toxicity test.

§ Percent mortality in 100% sample after 48 and 96 hours for *C. dubia* and *M. bahia*, respectively.

Methanol/water fractions were evaluated at 5 times and 2.5 times whole effluent concentration for *C. dubia* and *M. bahia*, respectively.

on the toxicity removed by filtration). Overall, the results showed that mysids were sensitive to the same non-polar organic toxicity as *C. dubia*. These tests confirmed non-polar organic toxicants as a cause of effluent toxicity to mysids.

Difficulties were encountered in trying to identify and confirm the specific non-polar organic toxicants. TIE Phase II procedures (USEPA, 1989b), which included HPLC separation and GC/MS analyses, tentatively identified more than 20 non-polar organic compounds as potential causes of toxicity. In addition, many potentially toxic unknown compounds were detected. The results suggested that the majority of the compounds were related to industrial sources because the compounds are not typically found in domestic wastewater. Further work was not performed to identify the toxic non-polar organic compounds because:

- Little or no toxicity data were available for most of the non-polar organic compounds identified in the effluent (e.g., no LC50 values for the specific non-polar organic compounds); therefore, it was not possible to determine if the concentrations present in the effluent were acutely toxic.

- The non-polar organic toxicants varied from sample to sample, which made it difficult to determine consistent causes of non-polar organic toxicity.
- Many of the compounds detected were unknowns.

The TIE results indicated that, in addition to ammonia, non-polar organic toxicity may need to be controlled to achieve compliance with the acute toxicity limit. Due to the difficulty in determining the non-polar organic toxicants, the LRSA decided to use a toxicity-based approach to identify the sources of non-polar organic toxicity and other non-ammonia effluent toxicity.

Toxicity Source Evaluation

The available information indicated that both ammonia and non-ammonia (e.g., non-polar organic) toxicity was being contributed by controllable industrial sources. Therefore, pretreatment controls were deemed to be feasible and source evaluation studies were performed to identify the sources of ammonia and non-ammonia toxicity. Sources of ammonia were identified by a chemical-specific approach and sources of non-ammonia toxicity were identified by a toxicity-based approach. The resulting information was used to develop appropriate pretreatment limits.

Chemical-Specific Source Evaluation

The LRSA conducted studies to locate the major sources of ammonia in the collection system. Key manholes and industrial discharges were sampled and tested for total ammonia from 1990 through 1992. The results indicated one major industrial source of ammonia in the collection system. Based on the survey results, the LRSA developed and implemented pretreatment limits to reduce effluent ammonia concentrations (LRSA, 1993a).

Toxicity-Based Source Evaluations

The toxicity-based approach used RTA procedures that involved treating industrial wastewater samples in bench-scale, batch simulations of the POTW activated sludge process and measuring the resulting toxicity (USEPA, 1989a). The toxicity remaining after batch treatment, referred to as “refractory” toxicity, represented the toxicity that passes through the POTW and causes effluent toxicity. As shown in Figure 5-2 (Section 5), two types of batch reactors are tested. A control reactor simulated the treatment plant and treated only the POTW influent. The second reactor evaluated the addition of the industrial discharge to the POTW by treating industrial wastewater spiked into the POTW influent. An industrial discharge would be considered a source of toxicity if effluent from the spiked reactor was more toxic than the control reactor effluent.

Initial RTA tests conducted during the USEPA study indicated that refractory toxicity was limited to an industrialized area of the collection system. Following the USEPA study, ammonia was confirmed as the primary cause of effluent toxicity and the major source of ammonia was identified. Accordingly, subsequent RTA tests focused on identifying sources of non-ammonia toxicity. In 1992, RTA testing was performed to evaluate sources of non-polar organic toxicity because non-polar organic compounds had been identified as a major cause of non-ammonia toxicity.

The procedure for measuring non-polar organic toxicity involved passing the RTA batch effluent samples through a C18 SPE column, eluting the column with methanol, and performing a toxicity test on the methanol elution (LRSA, 1992a). This procedure provided a direct means of measuring non-polar organic toxicity and it eliminated interferences associated with toxic ammonia concentrations

(i.e., ammonia was not captured by or eluted from the C18 SPE column).

The toxicity source evaluation identified two industrial dischargers of non-polar organic toxicity (LRSA, 1992b). Nonpolar organic toxicity tests performed on the effluent during this period suggested that non-polar organic toxicity was variable and that there may be other causes of non-ammonia toxicity. Therefore, further RTA testing was conducted in 1993 to identify sources of non-ammonia toxicity that may be caused by non-polar organic compounds and other unidentified compounds.

The ammonia pretreatment limits were not to become effective until after July 1995; therefore, the LRSA influent and effluent ammonia concentrations remained high during 1993. It was necessary to remove ammonia toxicity in RTA testing in order to identify sources of non-ammonia toxicity (LRSA, 1993b). Zeolite treatment of the batch effluent samples to remove ammonia was considered, but previous studies indicated that zeolite also may remove non-ammonia toxicity. Therefore, two alternative approaches were used to remove ammonia toxicity in the RTA. First, testing was conducted during periods of low influent ammonia concentrations, which occurred during the annual summer shutdown of the ammonia-contributing industrial process. During this period, ammonia concentrations were not acutely toxic; therefore, RTA testing would provide a direct measure of the non-ammonia toxicity contributed to the POTW. The second approach was used when the ammonia contributing process was fully operational and involved using a simulated plant influent (SPI). The SPI consisted of sewer wastewater collected from all major trunk lines except the sewer line serving the ammonia discharger. It was also necessary to wash the RAS used in the RTA to reduce the ammonia concentrations associated with the RAS (LRSA, 1993c).

The 1993 RTA testing was intended to identify those industries that would be required to meet pretreatment requirements to control non-ammonia toxicity. Thirty-two of the 40 industrial users were evaluated either directly or indirectly by testing sewer wastewater samples collected from key manholes. Previous RTA results and information obtained in an industrial user waste survey were used to select the industries to be tested.

The results of RTA tests performed in July and October 1993 are presented in Table G-2. If the effluent toxicity of the sewer wastewater spiked reactor was greater than that of the control reactor on two occasions, the discharge was considered a source of toxicity. Industries A, B, E, and F were indicated as sources of non-ammonia toxicity based on the results of direct testing of their industrial discharges. These

results support the findings of the USEPA study, which identified industries A, B, and E as sources of toxicity, and the 1992 study, which identified industries B and E as sources of non-polar organic toxicity. Six other industries were identified as suspected sources based on the results obtained for key manholes 9 and 12. LRSA plans to test these suspected sources directly to determine which industries are contributing toxicity.

Table G-2. Results of Refractory Toxicity Assessment, July and October 1993*

RTA Reactor Effluent	96-Hour Mysid TUa (100/LC50)						Source of Refractory Toxicity? ^ω
	Jul 15	Jul 16	Jul 22	Jul 23	Oct 19	Oct 20	
Control Reactor	<1.0	<1.0	1.63	1.05	2.0	1.75	n/a
Spiked Reactors							
Industry A	<1.0	NT	1.92	NT	3.39	1.22	YES
Industry B	1.45	NT	1.89	NT	NT	NT	YES
Industry C	<1.0	NT	NT	NT	NT	NT	NO
Industry D	NT	<1.0	NT	NT	NT	NT	NO
Industry E	NT	<1.0	NT	1.19	NT	1.75	YES
Industry E 5× †	NT	NT	NT	4.0	NT	NT	YES
Industry F	NT	<1.0	NT	2.18	1.55	1.86	YES
Industry G	NT	NT	<1.0	NT	NT	NT	NO
Industry H	NT	NT	NT	NT	2.28	NT	NO
Industry I	NT	NT	NT	NT	NT	1.29	NO
Industry J	NT	NT	NT	NT	NT	1.81	NO
Key manhole 1	<1.0	NT	NT	NT	NT	NT	NO
Key manhole 3	NT	NT	NT	1.12	‡	NT	NO
Key manhole 4	NT	<1.0	NT	NT	NT	NT	NO
Key manhole 7A	<1.0	NT	NT	<1.0	NT	NT	NO
Key manhole 9 §	1.1	NT	NT	<1.0	6.1	NT	YES
Key manhole 10	NT	NT	1.33	NT	NT	NT	NO
Key manhole 12 #	1.33	NT	1.81	NT	1.71	1.63	YES
Key manhole 14	NT	NT	1.33	NT	NT	NT	NO
Key manhole 15	NT	<1.0	NT	NT	NT	NT	NO
Roselle flume	NT	<1.0	NT	NT	NT	NT	NO

* Spiked reactor results shown in **bold** indicate greater TUa than the control. Increased toxicity in the spiked reactor effluent compared to the control indicates a source of refractory toxicity.

† Tested at five times the normal flow contribution to evaluate anticipated increase in flow.

‡ Toxicity test was invalid based on unacceptable control survival.

§ Key manhole 9 receives wastewater from three industries.

Key manhole 12 receives wastewater from three industries.

ω If a spiked reactor result was greater than that of the control on two occasions then the discharge was considered a source of refractory toxicity.

NT Not tested.

Toxicity Control Evaluation

The LRSA evaluated control options for ammonia and non-ammonia toxicants. The objective was to identify and assess the available options and to determine the most cost effective and pragmatic approaches for reducing effluent toxicity to acceptable levels.

Ammonia Toxicity Control Evaluation

A modified acute toxicity test procedure was developed by the LRSA and approved by the NJDEP to control pH drift in the toxicity test. The pH in previous LRSA compliance tests typically drifted up to 8.0 to 8.5, which resulted in an overestimation of ammonia toxicity (i.e., unionized ammonia concentrations increase as pH increases). The modified test procedure maintains pH in the toxicity test at the receiving system pH of 7.4. This modification provides a more accurate measurement of instream ammonia toxicity.

Using ammonia toxicity values for mysids published by USEPA (1989d), a linear regression model was prepared to predict the concentration of ammonia in the effluent which, in the absence of other toxicants, should result in compliance with the acute toxicity limit. The ammonia value generated by the model accounts for toxicity test conditions that affect the concentration of unionized ammonia (e.g., pH, temperature, and salinity). The model determined that the acute toxicity limit could be met with an effluent ammonia concentration of 35 mg/L (LRSA, 1991).

Several options for in-plant treatment of ammonia were evaluated to achieve the ammonia target level. As shown in Table 6-1 (Section 6), none of the six options evaluated was practical based on technical and cost considerations. In addition, significant inhibition of nitrification was observed during treatability tests, indicating that inhibitory compounds would need to be controlled if nitrification was selected as a control option (LRSA, 1991). Based on these results and the results of the ammonia source evaluation, chemical-specific pretreatment limits were selected as the best approach for controlling toxicity caused by ammonia (LRSA, 1993a).

Non-Ammonia Toxicity Control Evaluation

The TIE indicated that the causes of non-ammonia toxicity were complex and highly variable and the specific compounds causing non-ammonia toxicity could not be identified and confirmed. Consequently,

the necessary information was not available to develop chemical-specific pretreatment limits.

As an alternative to pretreatment limits, activated carbon treatment at the POTW was evaluated based on its effectiveness in reducing effluent toxicity caused by a variety of compounds including non-polar organic toxicants. Both PAC and GAC treatment were considered and found to be cost prohibitive (T.L. Morris, Technical Memorandum to LRSA, *Evaluation of Granular Activated Carbon at LRSA*, January 19, 1993). It also was determined that the use of PAC treatment would result in unacceptable sludge quality.

The LRSA elected to implement pretreatment controls because controllable industrial sources of non-ammonia toxicity had been identified and practical in-plant treatment options were not available. It was determined that the pretreatment limits must be toxicity-based because of the lack of specific information on the causes of non-ammonia toxicity. The proposed pretreatment approach involved RTA testing to determine which industries should be issued limits and which industries should be monitored to assess the need for future limits (LRSA, 1993c).

Implementation Of Toxicity Controls

Ammonia Pretreatment Limits

The approach used to develop pretreatment limits for ammonia was relatively straightforward. As required by the ACO, the LRSA submitted a work plan for developing ammonia pretreatment limits to the NJDEP in April 1992 and the plan was approved in May 1992 (LRSA, 1992c). Using the target ammonia level of 35 mg/L and the ammonia survey data, an allowable headworks loading approach (USEPA, 1987) was followed to develop draft pretreatment limits. The LRSA published the draft limits for public notice and comments were received and reviewed. In January 1993, the proposed ammonia pretreatment limits and the LRSA's response to public comments were submitted to the NJDEP. The limits were approved in March 1993 and industrial users were to comply with the limits by July 1995 (LRSA, 1993a).

Toxicity-Based Pretreatment Limits for Non-Ammonia Toxicity

The LRSA is one of the first municipalities to develop toxicity-based pretreatment limits to control non-ammonia toxicity. At the time of this study, toxicity-based pretreatment limits had not been applied

elsewhere and there was no specific guidance on developing such limits. The selected approach was based on the available TRE information and involved several aspects of various pretreatment approaches recommended by USEPA (1987).

The LRSA submitted a work plan for development of the limits to the NJDEP in June 1993 (LRSA, 1993b). The proposed approach was designed to address both major and minor sources of non-ammonia toxicity (LRSA, 1993c) and to ensure compliance without unnecessary controls. The proposed limits will consist of the following components referred to collectively as a toxicity management program (TMP):

- Narrative local pretreatment limit of “no discharge of refractory toxicity.”
- Pass/fail toxicity-based limit using the RTA procedure as a compliance test (i.e., the effluent LC50 of the industrial user spiked reactor may not be less than the LC50 of the control reactor effluent).
- Industrial user (if toxicity is found) may be required to implement a toxicity reduction program comprising requirements to identify causes and sources of toxicity, implement industrial user management practices, and evaluate and establish other controls to ensure compliance with the toxicity-based limits.
- RTA monitoring requirements and decision criteria for determining if an industrial user needs to continue with the TMP.
- Provisions to allow industries to be relieved from the TMP requirements if toxicity requirements are met.
- Compliance schedule including milestones and progress reports.
- Reopener clause stating that the pretreatment permit will be modified to include chemical-specific limits if the causes of toxicity are identified.

The proposed pretreatment limit approach falls under the case-by-case/best professional judgment approach described by USEPA (1987), but also includes toxicity-based requirements, industrial user management practice, and chemical-specific components. The TMP approach is consistent with USEPA recommendations for monitoring and controlling effluent toxicity through the NPDES.

The RTA procedures had not been used for compliance monitoring purposes in New Jersey. Therefore, a site-specific RTA protocol (LRSA, 1994) was submitted to the NJDEP for review and approval prior to development of the draft pretreatment limits. The RTA protocol was approved by the NJDEP in June 1996. Pretreatment program permits for several industries were modified to include the TMP provisions. These industries are currently required to conduct quarterly monitoring using the RTA protocol.

Discussion

Chemical-specific pretreatment limits are being implemented to control toxicity caused by ammonia and toxicity-based pretreatment limits are in place to control non-ammonia toxicity. The major source of ammonia ceased its discharge of the ammonia-laden waste stream in 1997. As a result, effluent ammonia concentrations at the LRSA treatment plant decreased to about 30 mg/L. A compliance test performed after the ammonia source was eliminated showed improved effluent quality (i.e., LC50 = 72%). Additional tests are planned to confirm this initial result.

It is possible that the ammonia pretreatment limits alone will achieve compliance with the acute effluent toxicity limit. However, due to the complex and variable nature of the non-ammonia toxicity, it is not possible to accurately predict if the ammonia reduction will achieve consistent compliance with the permit limit LC50 \geq 50%). The LRSA has established pretreatment requirements for non-ammonia toxicity to ensure full and timely compliance with the toxicity limit. The need for industrial users to control non-ammonia toxicity is ultimately tied to compliance with the acute effluent toxicity limit. If necessary, industrial users may request relief from these requirements if the effluent consistently complies with the acute effluent toxicity limit.

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